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A Review of Pseudorabies (Aujeszky's Disease) in Pigs

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SUMMARY

The main features in terms of etiology, clinical signs, pathogenesis, pathology, diagnosis, epizootiology and control and prevention that are known about pseudorabies are briefly reviewed. Areas still lacking in information which may provide more effective and reliable means of dealing with pseudorabies are mentioned and recommendations using available tools and knowledge to deal with the problem of pseudorabies are given.

RÉSUMÉ

Une revue de la pseudo-rage (maladie d'Aujeszky) chez le porc

Les auteurs présentent une brève revue des données actuellement connues et relatives aux aspects suivants de la pseudo-rage: étiologie, signes cliniques, pathogénèse, pathologie, diagnostic, épizootiologie, contrôle et prévention. Ils mentionnent certains autres aspects moins bien connus de cette maladie, qui pourraient peut-être nous aider à lutter plus efficacement contre elle. Ils donnent aussi des directives basées sur les connaissances actuelles, relatives à cette maladie.

INTRODUCTION

Pseudorabies (PR) is caused by *Herpesvirus suis* (porcine herpesvirus type 1) and has been endemic in various parts of Europe, particularly in the Central and Eastern regions, since Aladar Aujeszky first described and reproduced the disease in Hungary in 1902 (4). Hanson (26) referred to the description of a disease in the U.S. in 1813 which resembles PR but it was only in 1931 that Shope (59, 60) identified "mad-itch," as it was known in the U.S., as the same as Aujeszky's disease. It has since become known as pseudorabies because of some clinical resemblance to rabies.

Prior to the late 1960's, PR in the U.S. was seen as a sporadic disease but in recent years its incidence has been increasing steadily. The U.S. Department of Agriculture recorded 125 cases in 1974, 255 in 1975, 714 in 1976 and 1256 in 1977.

Canada is in a fortunate but precarious situation since PR has not yet been reported even though there is a large population of susceptible pigs. This paper is not intended as an extensive review of the literature on PR but as an outline of the current state of knowledge of PR to increase awareness of the disease. Thorough reviews of the literature have been published by Galloway (22) and Baskerville *et al* (12) and a chronological list of references has been prepared by Ryu (56).

Etiology

The virus causing PR has been assigned the binomial *Herpesvirus suis* (35) and details of the properties of the virus which is also known as porcine herpesvirus type 1, were reviewed by Kaplan (34) who described it as an enveloped DNA virus which has a wide cell culture host range and grows in chick embryos.

Clinical Features

Pseudorabies virus (PRV) causes clinical disease in all domestic livestock and a wide range of wild animals (22, 24), but is of greatest significance in swine. In most nonporcine species, all age groups are susceptible. The disease is manifested by various degrees of nervous disorder accompanied frequently by intense pruritus, usually at the primary site of infection often with respiratory signs of rhinitis and/or pneumonia. It is almost always fatal.

In pigs the clinical disease varies depending upon age of the pig, strain of virus involved and previous exposure. Pigs up to about four to five weeks of age are most susceptible and it is most severe, with a case fatality rate of up to 100% in piglets up to two weeks of age. Fatality rates

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progressively decrease as the age of affected animals increases. Some strains of PRV have however been associated with mortality in older pigs (12, 30).

Clinical signs of nervous derangement include muscular trembling, incoordination, ataxia, posterior paresis, opisthotonus, epileptiform convulsions, head pressing, circling motion and jaw champing (42). Pruritus is rarely seen in pigs (31) and blindness has been reported (30, 41).

Some strains of PRV are capable of producing a respiratory disease resulting in coughing, sneezing, nasal and ocular discharge and dyspnoea (11).

A common sequel to infection of pregnant sows is the occurrence of abortions, stillborn piglets and mummified or macerated fetuses, abortion usually takes place ten to 20 days after the onset of clinical illness (23, 44).

Pathogenesis

Experimentally PR can be produced by all routes of inoculation but the natural route is thought to be through nasal infection (24, 59). Field and Hill (21) considered primary viral multiplication at the initial site of infection to be important in relation to establishment of the disease in the host.

Following primary viral multiplication in the cells of the nasopharyngeal mucosa, the virus gains entry into the central nervous system (CNS) via various cranial nerves (49). Virus transfer along nerve fibres takes place within the axoplasm (45) and through Schwann's cells and fibroblasts of the endoneurium (16). Other pathways of viral dissemination throughout the body include the lymphatics (55, 67, 70) and further viral multiplication occurs in lymph nodes (32) and the vascular system into which the virus particles are believed to be carried by phagocytes (6). Pseudorabies virus has been found to proliferate in capillary endothelium, ganglion cells, satellite cells, Schwann's cells (14), lymphocytes and macrophages (16).

Abortion, stillbirth and mummification are well recognized features of PR but the mode of fetal infection is still not fully understood.

Pathology

Gross changes are negligible. The brain may present various degrees of meningeal and cerebral vascular congestion, accompanied by edema and excessive cerebrospinal fluid (20, 24). Some strains of PRV produce mild to severe inflammatory and necrotic changes in the mucosa of the upper respiratory tract (11, 18, 44) and lungs (7, 8, 9, 10). Focal necrosis in the liver, spleen, tonsils, lungs (36) and cranial, cervical and bronchial lymph nodes (6, 31, 49) are not uncommon.

Microscopically, the main lesion in the CNS is a nonsuppurative meningoencephalomyelitis with neuronal degeneration and necrosis, neuronophagia, diffuse and focal gliosis and perivascular cuffing by mononuclear cells (12, 24). Inflammation and necrosis in the respiratory passages are unselective and may involve all divisions of the respiratory "tree" (8, 9, 10, 11). Intranuclear inclusions may be found in neurones, astrocytes, oligodendroglia, Purkinje cells (20), respiratory epithelium, pulmonary macrophages (8) and lymph node macrophages (24) and are of diagnostic significance when found.

Diagnosis

Clinically PR in swine bears resemblance to a wide range of diseases but the occurrence of reproductive problems in gilts and sows accompanied by neurological signs in growing, especially suckling pigs, is highly suggestive of PR. The pathological findings may provide additional evidence and although not frequently found, intranuclear inclusions are regarded as highly significant diagnostically.

Laboratory tests are essential for a definitive diagnosis. Fluorescent antibody tests to detect PRV in tissue (57) and in tissue culture (52, 65) have been used. The preferred tissue specimens for these tests include fresh samples of tonsils, olfactory bulb, pons, nasal mucosa, cervical lymph nodes and the root of the trigeminal nerve.

Virus isolation can be carried out in a wide range of tissue culture systems (34). Lee (42) isolated PRV most frequently from olfactory bulb, pons, cerebellum and the medulla and less frequently from the nasal mucosa, retropharyngeal lymph nodes, tonsils, lungs and bronchial lymph nodes.

Although general serological tests are available, the most commonly used procedure is the serum neutralization test for which paired serum samples are required.

Cutaneous allergic tests have been utilized (63) and may be a useful aid in the detection of chronic virus carriers.

Prior to the availability of tissue cultures, laboratory animals, particularly rabbits, were used for diagnostic purposes. Subcutaneous inoculation of infective material leads to intense pruritus (average 40-50 hours later) followed by death (12).

Epizootiology

Survival of PRV in the environment under various conditions has been studied (3, 54, 64, 71, 72). Under suitable conditions it has been found to survive in hay, wood and food for up to 46 days and thus infection from a contaminated environment is possible.

Under natural conditions, the proximity of susceptible animals and infected animals both within and between species is important, as ingestion or inhalation of infected material may result in disease (59, 60).

Virus recovery from feces and urine have been reported (13, 19, 37, 53). However, McFerran and Dow (47, 48, 49) and Sabo *et al* (58) were unable to demonstrate virus in feces and urine.

Transmammary spread of PRV from infected

sows to their offspring was demonstrated by Kojnok (37), who isolated the virus from the milk of six of 15 symptomless sows. Coital transmission was postulated by Akkermans (1) who recovered virus from the prepuce and vagina of infected animals.

Rats have long been implicated as an important agent in the transmission of PR and Shope (60) postulated transmission may be achieved when infected rats were eaten by pigs (or other species). Aldasy and Mate (2), however, concluded that infected rats did not excrete the virus and therefore played only a minor role in the spread of PR. Kanitz (33) presented experimental evidence of lateral spread of virus between pigs and raccoons, pigs and opposums and vice versa. Since infected rats and wild animals can travel some distance before dying, geographical spread of PR may be attributed to these species in some cases.

However, it is now generally accepted that the pig is the main reservoir of PRV and that carrier states do exist. Kojnok (38) has demonstrated virus from swine up to six months after recovery. Howarth and De Paoli (30) and Howarth (28) noted some outbreaks to coincide with environmental extremes and suggested that latent carriers do not shed virus unless placed under stress. Transportation over long distances, concurrent disease, overcrowding and possibly also parturition may provide such stress situations.

Control and Prevention

For a long time the only means of control of and prevention against PR was through hygiene measures and test and slaughter policies. In many areas around the world especially where the disease occurs sporadically this is still the recommended approach. No successful therapy is available.

In parts of Eastern and Central Europe where PR is endemic and often occurs in epizootic proportions, research has been centred around various vaccines, both killed and modified live virus vaccines, a long list of which appear in the literature (12, 42, 56). Some have been shown to be ineffective but some have been in use successfully in several European countries. According to Skoda (61), the incidence of the disease in those countries did not decrease although economic losses were reduced.

Some of the more noteworthy modified live vaccines include a Bucharest strain grown in chicken embryos with $Al(OH)_3$ adjuvant (17), the K-variant (or Bartha) strain propagated in chicken embryo fibroblasts (CEF) or calf testicle culture (5, 39), the Bartha strain grown in Vero cells (51) and the "BUK" strain grown in CEF (62), chicken embryo (66) and pig kidney cells (29).

There are two commercially available vaccines in the United States of America. A live vaccine (Norden Laboratories) originally of the "BUK" strain but since passaged in pig kidney and modified (68) and an inactivated vaccine (Salsbury Laboratories). An intramuscular dose of 1 ml gave up to 100% protection against experimental intranasal challenge. However, the live vaccine is only recommended for use in pigs as it causes disease in other species (68).

Killed vaccines have had the reputation of being ineffective although Baskerville *et al* (12) mentioned several authors who have reported successful protection of various species under experimental conditions. There are two noteworthy killed vaccines used in Europe; namely a Roumanian vaccine which contained a field isolate (designated "B.C.") propagated on pig kidney cell cultures, inactivated with saponin with an $Al(OH)_3$ adjuvant (15) and a French vaccine prepared from a field isolate, propagated on the cell line IBR's$_2$, inactivated by formaldehyde and mixed in an oil adjuvant (69). The $Al(OH)_3$ adjuvant vaccine gave up to 85% protection (15) and the oil adjuvant vaccine gave up to 100% protection against experimental challenge and 95% of piglets from vaccinated sows withstood experimental challenge (69).

Using the Aujeszky strain, Canadian studies on a formalin killed vaccine by Lee and Wilson (43) gave up to 95.5% protection against experimental intranasal challenge which produced clinical signs in 100% and death in 38% of control pigs.

Passively acquired maternal antibodies via colostrum have been shown to protect piglets against death (69) but the piglets are still susceptible to infection (40). McFerran and Dow (50) suggest that the level of protection conferred upon piglets by colostral antibodies may be dependent upon the amount of antibody transferred from sow to piglets, but piglets protected by colostral antibodies were found to excrete virus up to 20 days following challenge and may thus obscure the presence of the disease and contribute to further spread of the virus.

When faced with an imminent exposure, the use of antiserum intravenously or intraperitoneally has prophylactic value but is of little value therapeutically (25, 46). Whole serum (from swine or horses) may be used but gammaglobulin prepared from swine PR-antiserum provides better protection than gammaglobulin from equine PR-antiserum. Hill and Glock (27) demonstrated protection against intranasal challenge in piglets given 5 ml swine PR-antiserum subcutaneously. Owing to the cost of antiserum and gammaglobulin preparation and their limited application, their use in control and prevention is impractical.

CONCLUSION

Much information on many aspects of PR is known and available but certain key areas remain largely unexplored. Thus more information is needed regarding the immunological basis of resistance against PR; the circumstances and factors pertaining to the carrier state and the means of identifying the carrier animal and to

distinguish between the vaccinated animal and that which have had a natural infection. Until these features of PR are understood we can merely learn to live with the disease if we happen to be in an endemically infected area, employing vaccines to reduce death losses and relying on zoosanitary measures to contain the spread of the disease. In PR free areas there must be constant and continuous surveillance of incoming livestock, pigs in particular, against the introduction of the virus. Serological surveys, quarantine and the restriction of introduction of animals must be practiced with vigilance.

REFERENCES

1. AKKERMANS, J.P.W.M. Aujeszky's disease in swine in the Netherlands. Rotterdam: Central Diergeneeskundig Instituit. Abstracted in Vet. Bull. 33: 497. 1963.
2. ALDASY, P. and L. MATE. Aujeszky's disease and the brown rat. Magy. Allatorv. Lap. 24:324–326. 1969.
3. ATANASOVA, KH. Resistance of Aujeszky's disease virus in the urine of pigs and sheep. VetMed. Nauki, Sof. 9: 69–74. 1972.
4. AUJESZKY, A. Ueber eine new Infektions krankheit bei Haustieren. Zentbl. Bakt. ParasitKde I 32: 353-357. 1902.
5. BARTHA, A. and J. KOJNOK. Active immunization against Aujeszky's disease. pp. 531–533. Proc. 17th Int. Vet. Cong., Hanover. 1963.
6. BASKERVILLE, A. A study of the pulmonary tissue of the pig and its reaction to the virus of Aujeszky's disease. Ph.D. Thesis, Queen's University, Belfast. 1971.
7. BASKERVILLE, A. The histopathology of pneumonia produced by aerosol infection of pigs with a strain of Aujeszky's disease virus. Res. vet. Sci. 12: 590–592. 1971.
8. BASKERVILLE, A. Ultrastructural changes in the pulmonary airways of pigs infected with a strain of Aujeszky's disease virus. Res. vet. Sci. 13: 127–132. 1972.
9. BASKERVILLE, A. The histopathology of experimental pneumonia in pigs produced by Aujeszky's disease virus. Res. vet. Sci. 14: 223–228. 1973.
10. BASKERVILLE, A. Ultrastructural changes in the lungs of pigs affected with Aujeszky's disease virus. Res. vet. Sci. 14: 229–233. 1973.
11. BASKERVILLE, A., R.M. MCCRACKEN and J.B. MCFERRAN. The histopathology of experimental rhinitis in pigs produced by a strain of Aujeszky's disease virus. Res. vet. Sci. 12: 323–326. 1971.
12. BASKERVILLE, A., J.B. MCFERRAN and C. DOW. Aujeszky's disease in pigs. Vet. Bull. 43: 465–480. 1973.
13. BASS, E.P., R.L. SHARPEE and E.T. THURBER. Immunization for pseudorabies — cost effective protection against Aujeszky's disease. Proc. Am. Ass. swine Practnrs, Kansas City, Missouri. March 1978.
14. BECKER, C.H. (Multiplication of Aujeszky's virus in the spinal ganglia of the rabbit). Arch. exp. VetMed. 22: 363–381. 1968.
15. BERBINSCHI, C. Roumanian inactivated vaccine against Aujeszky's disease. Cah. Méd. vét. 43: 324–329. 1974.
16. BLASKOVIC, D., A. SABO and J. RAJCANI. (Experimental pathogenesis of Aujeszky's disease in piglets). Arch. exp. VetMed. 24: 9–27. 1970.
17. BRAN, L., I. SUHACI and R. URSACHE. Prophylaxis of Aujeszky's disease. Archiva vet. 1: 19–35. 1965.
18. BRAN, L., I. SUHACI and R. URSACHE. Experimental production of Aujeszky's disease by nasal instillation of culture virus. Archiva vet. 5: 83–97. 1968.
19. CORNER, A.H. Pathology of Aujeszky's disease in piglets. Res. vet. Sci. 6: 337–342. 1965.
20. DOW, C. and J.B. MCFERRAN. The neuropathology of Aujeszky's disease in the pig. Res. vet. Sci. 3: 436–468. 1962.
21. FIELD, H.J. and T.J. HILL. The pathogenesis of pseudorabies in mice: virus multiplication at the inoculation site and axonal uptake. J. gen. Virol. 26: 145–148. 1975.
22. GALLOWAY, J.A. Aujeszky's disease. Vet. Rec. 50: 745–762. 1938.
23. GORDON, W.M. and D. LUKE. An outbreak of Aujeszky's disease in swine with heavy mortality in piglets, illness in sows and death in uterus. Vet. Rec. 67: 591–597. 1955.
24. GUSTAFSON, D.P. Pseudorabies. *In* Diseases of Swine. 4th Ed. pp. 391–410. H.W. Dunne and A.D. Leman, Eds. Ames, Iowa: Iowa State University Press. 1975.
25. GUSTAFSON, D.P., C.L. KANITZ and M.V. MITCHELL. Immunologic approaches to the control of pseudorabies. J. Am. vet. med. Ass. 160: 623–628. 1972.
26. HANSON, R.P. The history of pseudorabies in the United States. J. Am. vet. med. Ass. 124: 259-261. 1954.
27. HILL, H.T. and R.D. GLOCK. The efficacy of pseudorabies antiserum prophylaxis in suckling pigs. p. G.7. Proc. 4th Int. Pig Vet. Soc. 1976 Cong. Ames, Iowa. June 1976.
28. HOWARTH, J.A. A serologic study of pseudorabies in swine. J. Am. vet. med. Ass. 154: 1583–1589. 1969.
29. HOWARTH, J.A. Response of swine to virulent and modified pseudorabies viruses. pp. 371–384. Proc. 74th a. Meet. U.S. Anim. Hlth Ass. 1970.
30. HOWARTH, J.A. and A. DE PAOLI. An enzootic of pseudorabies in swine in California. J. Am. vet. med. Ass. 152: 114–118. 1968.
31. HURST, E.W. Studies of pseudorabies (infectious bulbar paralysis, mad itch). I. Histology of the disease with a note on symptomatology. J. exp. Med. 58: 415–433. 1933.
32. JAMRICHOVA, O. and R. SKODA. Multiplication of pseudorabies virus in the inguinal lymph node of pigs. Acta. virol., Prague 12: 555. 1968.
33. KANITZ, C.L. The role of wild life and domestic animals. p. 29a. Pseudorabies Fact Finding Conference. Iowa State University, Ames, Iowa. April 1977.
34. KAPLAN, A.S. Herpes simplex and pseudorabies viruses. Virol. Monogr. 5: 26–27. 1969.
35. KAPLAN, A.S. The Herpesviruses. p. 456. New York: Academic Press. 1973.
36. KLUGE, J.P. and C.J. MARÉ. Gross and microscopic lesions of pre-natal and neo-natal pseudorabies (Aujeszky's disease) in swine. p. G.3. Proc. 4th Int. Pig Vet. Soc. 1976 Cong. Ames, Iowa. June 1976.
37. KOJNOK, J. Transmission of Aujeszky's disease in sow's milk. Magy. Allatorv. Lap. 12: 23–24. 1957.

38. KOJNOK, J. The role of carrier sows in the spreading of Aujeszky's disease to suckling pigs. Data on Aujeszky's virus carriership among fattening pigs. Acta vet. hung. 15: 283–295. 1965.
39. KOJNOK, J. and A. BARTHA. Immunization experiments with attenuated Aujeszky's disease virus. Magy. Allatorv. Lap. 17: 19–20. 1962.
40. KOJNOK, J. and J. SURJAN. Investigations concerning the colostral immunity of pigs in the cases of Aujeszky's disease. Acta vet. hung. 13: 111–118. 1963.
41. LAMONT, H.G. and P.L. SHANKS. An outbreak of Aujeszky's disease amongst pigs. Vet. Rec. 51: 1407–1408. 1939.
42. LEE, J.Y.S. The efficacy of an inactivated vaccine against pseudorabies (Aujeszky's disease) in pigs, sheep, cats and rabbits. M.Sc. Thesis, University of Guelph, Ontario. 1978.
43. LEE, J.Y.S. and M.R. WILSON. Immunization of swine against Aujeszky's disease with an inactivated adjuvanted vaccine. Proc. 5th Int. Pig Vet. Soc. Zagreb, Yugoslavia. 1978.
44. MARÉ, C.J., J.P. KLUGE and G.W. BERAN. Reproductive failure in swine infected with pseudorabies (Aujeszky's disease). p. G.1. Proc. 4th Int. Pig Vet. Soc. 1976 Cong. Ames, Iowa. June 1976.
45. MCCRACKEN, R.M., J.B. MCFERRAN and C. DOW. The neural spread of pseudorabies virus in calves. J. gen. Virol. 20: 17–28. 1973.
46. MCFERRAN, J.B. Comments on immunological approaches to the control of pseudorabies. J. Am. vet. med. Ass. 160: 629–630. 1972.
47. MCFERRAN, J.B. and C. DOW. Virus studies on experimental Aujeszky's disease in calves. J. comp. Path. 74: 173–179. 1964.
48. MCFERRAN, J.B. and C. DOW. The distribution of the virus of Aujeszky's disease in experimentally infected sheep. Res. vet. Sci. 5: 143–148. 1964.
49. MCFERRAN, J.B. and C. DOW. The distribution of virus of Aujeszky's disease (pseudorabies virus) in experimentally infected swine. Am. J. vet. Res. 26: 631–635. 1965.
50. MCFERRAN, J.B. and C. DOW. The effect of colostrum derived antibody on mortality and virus excretion following experimental infection of piglets with Aujeszky's disease virus. Res. vet. Sci. 15: 208–214. 1973.
51. MCFERRAN, J.B. and C. DOW. Studies on immunization of pigs with the Bartha strain of Aujeszky's disease virus. Res. vet. Sci. 19: 17–22. 1975.
52. MEYLING, A. and V. BITSCH. Fluorescent antibody diagnosis of pseudorabies. Acta vet. scand. 9: 360–368. 1967.
53. NIKITIN, M.G. Pigs as carriers of Aujeszky's disease virus. Veterinariya, Moscow No. 9. 32–36. 1961.
54. POLYAKOV, A.A. and YU. I. ANDRYUNIN. Disinfection of liquid manure contaminated with foot and mouth disease virus and Aujeszky's disease virus. Veterinariya, Moscow No. 5. 45–47. 1974.
55. RAJCANI, J. and A. SABO. (Histological and immunohistological studies on the pathogenesis of Aujeszky's disease in piglets). Zentbl. VetMed. 16B: 541–552. 1969.
56. RYU, E. Chronological reference of zoonoses, Aujeszky's disease, pseudorabies. Dept. of Vet. Med., National University, Taipei, Taiwan. 1975.
57. SABO, A. and J. RAJCANI. Rapid diagnosis of Aujeszky's disease by the fluorescent antibody technique. Acta Virol., Prague 14: 475–484. 1970.
58. SABO, A., J. RAJCANI and D. BLASKOVIC. Studies on the pathogenesis of Aujeszky's disease. III. The distribution of virulent virus in piglets after intranasal infection. Acta virol., Prague 13: 407–414. 1969.
59. SHOPE, R.E. A contagious disease in swine. Science 80: 102–103. 1934.
60. SHOPE, R.E. Experiments on the epidemiology of pseudorabies. I. Mode of transmission of the disease in swine and their possible role in its spread to cattle. II. Prevalence of the disease among Middle-West swine and the possible role of rats in herd-to-herd infections. J. exp. Med. 62: 85–99, 101–117. 1935.
61. SKODA, R. The control of Aujeszky's disease in large swine units by use of attenuated live vaccines. p. G.6. Proc. 4th Int. Pig Vet. Soc. 1976 Cong. Ames, Iowa. June 1976.
62. SKODA, R., I. BRAUNER, E. SADECKY and J. SOMOGIOVA. Immunization of Aujeszky's disease with live vaccine. II. Immunization of pigs under laboratory conditions. Acta virol., Prague 8: 123–134. 1964.
63. SMITH, P.C. and W.L. MENGELING. A skin test for pseudorabies virus infection in swine. Can. J. comp. Med. 41: 364–368. 1977.
64. SOLOMKIN, P.S. and M.I. TUTUSHIN. Viability of the virus of Aujeszky's disease in fodder, and on products of animal origin. Veterinariya, Moscow 33: 49–50. 1956.
65. STEWART, W.C., E.A. CABREY and J.I. KRESSE. Detection of pseudorabies virus by immunofluorescence. J. Am. vet med. Ass. 151: 747–751. 1967.
66. STOICHEV, S., S. NEDYALKOV and B. RIBAROV. Adsorbate vaccine against Aujeszky's disease. VetMed. Nauki, Sof. 2: 861–866. 1965.
67. TAGA, M., C. BERBINSCHI, I. CIRSTET and I. COMAN (Distribution of Aujeszky's disease virus in pigs experimentally infected by various routes). An. Inst. Pat. Igien. anim. 7: 221–228. 1957.
68. THURBER, E.T. A new modified live virus vaccine for prevention of pseudorabies. Proc. 18th Annual George A. Young Conference and 17th Annual Nebraska SPF Conference. Lincoln, Nebraska. August 1977.
69. TOMA, B., J.F. DELAGNEAU, R. LOQUERIE and P. PRUNET. A study of a new, inactivated Aujeszky's disease vaccine with adjuvant. Bull. Off. int. Epizoot. 84: 223–237. 1975.
70. TUNG, M.C., C.J. LIU, C.C. LEE and H.S. KWANG. Studies on swine pseudorabies. II. Viral distribution and histopathology in experimentally infected swine. J. Taiwan Ass. Anim. Husb. vet. Med. No. 25. 1–15. 1974.
71. USTENKO, V.S. Survival of the virus of Aujeszky's disease. Veterinariya, Moscow 34: 74–75. 1957.
72. USTENKO, V.S. Survival of the virus of Aujeszky's disease in the external environment. Trudy vses. Inst. vet. Sanit. 13: 49–59. 1958.